Title: Comment on Charlier et al., 2019: *"The Mandible of Saint-Louis (1270 AD): Retrospective Diagnosis and Circumstances of Death"*

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We read with interest the recent article by Charlier and colleagues entitled “The Mandible of Saint-Louis (1270 AD): Retrospective Diagnosis and Circumstances of Death”[1]. This work, which consisted of macroscopic examination of a mandible purported to belong to the Crusader King of France and a review of historical accounts pertaining to his death, generated a good deal of press coverage and stimulated public interest in the fascinating field of paleopathology. However, as researchers engaged in studying dental and nutritional disease in archaeological human remains we have some concerns with the methodology employed by the authors and the conclusions reached from the data they have collected. These include issues of provenance, lesion description, diagnostic methodology employed, and problems with historical references.

1.1 Problems with provenance and methods used for authentication

One of the stated aims of Charlier and colleagues was to confirm the authenticity of the mandible. Provenance is a widely acknowledged issue in bioarchaeological research, particularly in the case of venerated human remains[2]. It is known that the relics of Louis IX have been scattered and stored in different places throughout Europe since the Middle Ages[3]. From the 12th century, official authentication documents “authentique” accompanied the relics and allowed the traceability of their movements and modifications over time [4]. However, Charlier and colleagues[1] have not provided evidence of this original authentication with the mandible they examined. The authors state that “*..a perfect traceability of the remains is given by historical sources*” but provide no support for this assertion. The presence of a label on the mandible, which is purported to be an addition dated from the 18th century, is not sufficient to authenticate the mandible. Although this does not preclude the analysis the authors have conducted, the fact that the mandible cannot be truly authenticated should have been acknowledged.

There are also issues with the osteological methods used by Charlier et al.[1] to support their identification. The authors cite Parr et al. [5] to justify the age and sex assigned to this individual. However, Parr et al.’s [5] article focuses on variation in mandible morphology with age and does not provide standards for estimating age or sex. In fact, there are currently no osteological methods for estimating age from an edentulous mouth, as is the case with the mandible described. Even if dental attrition could have been employed, such methods are incapable of yielding the precise age of 56 years reported by Charlier et al. [1]. The authors employ a 3D methodology [6] to “face-match” the mandible against a statue of Saint Louis. This paper compares mortuary masks to the skull of known individuals and it is not appropriate to apply them to the remains of St. Louis as there is no guarantee that the statue is an accurate representation of the living king. This statue was produced at the beginning of the 14th century (not the 13th as stated in the paper) almost 40 years after the death of the king [7]. During this period it was usual to represent a previous king with the face of the ruling one [7] [8]. It is also not known if the statue is life-size. The mandible should have been scaled to fit the statue, but if this was performed it is not reported. Because the mandible lacks the temporomandibular articulations it is impossible to position the bone in relation to the rest of the face as depicted in Figure 3, rendering this superimposition essentially meaningless. Finally, a very broad radiocarbon range of ~300-400 years (when the marine reservoir effect is considered), which encompasses the life of Louis IX, is provided from the mandible. In short, although none of the results are necessarily inconsistent with an identification, they are either too imprecise or methodologically problematic to definitively support a positive one.

1.2 Problems with the description of oral lesions

Another methodological issue is the use of incorrect and/or inconsistent descriptions of the potential pathological lesions reported in the mandible. The paper appears to contradict itself with regard to the porosity visible on the alveolar margins of the mandible. The authors say

that there was "*no evidence of alveolar reabsorption* [*sic*]" and in the same paragraph that "*The jugal side of the bone, at the level of teeth 44, 45 and 46, showed a moderate to severe resorption, with a porosity of the alveolar bone consistent with inflammation and/or infection (Fig 2)*" [1]. Some porosity consistent with chronic inflammation is observable on the right alveolar crest documented in Figure 1, but without the teeth present it is very difficult to be sure of the level of alveolar resorption that has taken place, and whether the current porosity relates to an episode of gingivitis/periodontitis or a condition such as scurvy which causes gingival inflammation [9].

There are similar contradictions in the description of Figure 2 with their statement "*No related abscesses or any other lesions were visible on the CT-scan"* but the caption for Figure 2 details that "*3D rendering of Saint Louis lower jaw shows evidence of three adjacent and partially fused periodontal apical cysts/granuloma* (teeth 44, 45, 46). Periapical granulomas, periapical cysts and periapical abscesses are difficult to differentiate without microscopy and the blanket term 'alveolar lesions' is preferred [9]. The exact aetiology of the lesion types are varied, although they are most commonly caused by exposure or infection of the pulp. This may be from caries, advanced tooth wear, or trauma to the tooth. Many oral conditions are interrelated and identifying the cause of the alveolar lesion is not always clear. To identify the alveolar lesions that appear to be associated with teeth 44, 45 and 46, clearer photos and additional analyses would be necessary. From the photos, it is also difficult to identify the extent of postmortem damage as there appears to be some postmortem breaks present in the sockets of 44, 45, and 46 and in Figure 2 it is not obvious which regions the authors believe are affected by alveolar lesions vs. normal anatomy (i.e. the mental foramen). Radiography or microCT would be more helpful here than a 3D reconstruction as it would allow visualisation of the internal structures of the mandible.

1.3 Lack of differential diagnosis or use of established diagnostic methods

Differential diagnosis, the process of successive exclusion of less likely aetiologies for the lesions observed, is an essential component of any case study in clinical medicine or palaeopathology. The authors have not demonstrated this process in the text or in any supplementary data and as a result their diagnosis of scurvy does not have a firm foundation. Scurvy is a condition caused by prolonged and severe deficiency of ascorbic acid, a vitamin necessary for a number of physiological processes including the formation of collagen [10]. Scurvy, and the process of recovery from the disease, can result in a number of characteristic skeletal lesions including fine abnormal cortical porosity, subperiosteal new bone (representing healing haematomas), and a number of endochondral defects in actively growing infants and children [11][12]. Not all of these changes are equally associated with scurvy and none of them, with the exception of some endochondral lesions, are unique to the disease. Because of this, differential diagnosis should include a number of other infectious and metabolic conditions and a suite of lesions in multiple skeletal elements is required for a diagnosis of probable scurvy to be reached [13]. An extensive methodological body of work on the skeletal lesions of scurvy exists, none of which are employed or acknowledged by Charlier and colleagues [1], who elect instead to cite in the discussion a short commentary on the clinical presentation of the disease and an editorial that is primarily concerned with the use of ascorbic acid as a sepsis treatment. Work on the skeletal lesions of scurvy was first published by Barlow [14] more than a century ago and expanded upon by many researchers using a synthesis of clinical, anthropological, and theoretical pathophysiological sources [15] [13]. Although various diagnostic schemes have been proposed, there is a general consensus among the palaeopathological community that no single lesion allows identification of the condition and the most strongly diagnostic lesions are associated with the chronic haemorrhage caused by compromised vascular integrity. Periodontal disease and antemortem tooth loss can be associated with scurvy due to chronic gingival haemorrhage and weakening of the alveolar ligament [16], but are not diagnostic. Moreover, Charlier and colleagues[1] contradict their own logic in using these features to support a diagnosis of scurvy which they allege occurred during the *7th crusade*.

The alveolar lesions depicted in Figures 1 and 2 are clearly either active or in the early stages of remodelling. These *could not* have occurred 12 years prior to the death of Louis IX. In short, the skeletal lesions upon which the authors' diagnosis of scurvy are ambiguous and chronologically unsupported by historical accounts of the 7th crusade.

1.4 Issues with historical references

Finally, the authors make some use of some historical sources to support their diagnostic claims but there are issues here as well. Charlier et al.[1] state that a nobleman, Jean de Joinville, who was present during the 7th crusade with the king, noted widespread trench disease (*la maladie de l'ost*). They do not directly link the pathology noted for the mandible with "trench mouth" or (acute) necrotising ulcerative gingivitis, a common problem caused by a normal commensal bacteria during periods of compromised immunity, for example in the soldiers in the trenches during the 1914-18 war in Europe, or modern adults with HIV infections [17]. However, to support their claims of scurvy, they "*speculate that an epidemic of trench mouth coupled with a major epidemic of epidemic typhus (Rickettsia prowazekii) and/or trench fever (Bartonella quintana) may also have occurred as co-infection may be frequent with such chronic vitamin deficiency"*. No historical or archaeological contextual information on the dietary habits of the French campaigners is provided, other than a general statement that scurvy has been noted in other military campaigns throughout history. It is certainly possible that during the 7th crusade the French suffered from trench mouth, gingival lesions due to scurvy, or a combination of both. However, it is not known if this description is relevant to conditions experienced by soldiers during the 8th crusade when Louis IX died or indeed if the King was living in conditions that would have placed him at the same risk for these conditions as common soldiers.

Although historical sources can be beneficial in cases such as this, there needs to be careful consideration of their use in retrospective diagnosis. Descriptions of disease in historical texts need to first be examined for observer bias, translation errors, recognition of advancement of modern medicine, and the cultural context of the written source, which can all result in incorrect attribution of the symptoms described in the historical text to modern disease diagnosis [18]. Historical sources, like skeletal lesions, require critical evaluation and cannot be accepted as infallible.

2. Conclusions

Paleopathology is truly an interdisciplinary field to which clinicians and specialists in other areas contribute significantly. However, a familiarity with established anthropological methods and an acknowledgment of the limitations inherent in the study of disease in skeletal and dental remains is vital for accurate interpretations. Failure to engage with these topics reflects poorly on our discipline and can lead to unrealistic expectations from the public about the meaning of data collected from human skeletal remains.

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